ILE 'MEDLINE' ENTERED AT 17:48:28 ON 11 DEC 2006

FILE 'BIOSIS' ENTERED AT 17:48:28 ON 11 DEC 2006

=> s nogo
L1 26531 NOGO

=> s caspr
L2 161 CASPR

=> s l1 and l2
L3 4 L1 AND L2

=> disp l3 ibib abs 1-4

L3 ANSWER 1 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2004241488 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15139292
TITLE: New insights on neuronal alterations in jimpy mutant brain.
AUTHOR: Harsan Laura; Jalabi Walid; Grucker Daniel; Ghandour M Said
CORPORATE SOURCE: UMR 7004 CNRS/ULP, Institut de Physique Biologique, Faculte de Medecine, 11 rue Humann, 67085 Strasbourg, France.
SOURCE: Neurochemical research, (2004 May) Vol. 29, No. 5, pp. 943-52.
Journal code: 7613461. ISSN: 0364-3190.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 14 May 2004
Last Updated on STN: 28 Jul 2004
Entered Medline: 27 Jul 2004

AB In spite of abundant data on oligodendrocyte abnormalities in dysmyelinated jimpy brain, little is known about the axonal damage and the expression of neuronal genes. Recent findings indicate that Nogo -A, oligodendrocyte-myelin glycoprotein (OMgp), and myelin-associated glycoprotein (MAG) inhibit axonal growth by binding a common receptor, the Nogo-A receptor (NgR)-p75 complex. In order to evaluate neuronal modifications in the absence of myelin and in the presence of abnormal oligodendrocytes at different developmental stages, the expression of these inhibitory proteins and their receptors was investigated in jimpy mutant brain. Despite the decrease in oligodendrocyte number at P15 and P25 in jimpy, Nogo-A and OMgp mRNA levels are not significantly different compared with control, suggesting an overexpression of neuronal Nogo-A and OMgp in mutant. Double immunolabeling for Nogo -A and neurofilaments shows strong axonal staining of Nogo-A in jimpy and its down-regulation in oligodendrocytes. The current data raise questions about functions of Nogo-A other than neurite growth inhibition in the CNS. No significant changes in NgR mRNA levels were observed in jimpy, where the increase in p75 level can be correlated with the cell death of oligodendrocytes. In the paranodal region, the cell adhesion molecule neurofascin glial isoform NFN155 mRNA level is reduced by 40% whereas neuronal form NFN186 is up-regulated. These results may explain the failure of paranodal region organization, even with normal level of CASPR (paranodin) mRNA detected in jimpy brain.

L3 ANSWER 2 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2003549087 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14592966
TITLE: Nogo-A at CNS paranodes is a ligand of Caspr: possible regulation of K(+) channel localization.

Case # 537757
STN (MEDLINE, BIOSIS)
12/11/06 AA

AUTHOR: Nie Du-Yu; Zhou Zhi-Hong; Ang Beng-Ti; Teng Felicia Y H; Xu Gang; Xiang Tao; Wang Chao-Yang; Zeng Li; Takeda Yasuo; Xu Tian-Le; Ng Yee-Kong; Faivre-Sarrailh Catherine; Popko Brian; Ling Eng-Ang; Schachner Melitta; Watanabe Kazutada; Pallen Catherine J; Tang Bor Luen; Xiao Zhi-Cheng
CORPORATE SOURCE: Department of Clinical Research, Singapore General Hospital, Singapore.
CONTRACT NUMBER: NS27336 (NINDS)
SOURCE: The EMBO journal, (2003 Nov 3) Vol. 22, No. 21, pp. 5666-78.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 22 Nov 2003
Last Updated on STN: 17 Dec 2003
Entered Medline: 16 Dec 2003

AB We report Nogo-A as an oligodendroglial component congregating and interacting with the Caspr-F3 complex at paranodes. However, its receptor Nogo-66 receptor (NgR) does not segregate to specific axonal domains. CHO cells cotransfected with Caspr and F3, but not with F3 alone, bound specifically to substrates coated with Nogo-66 peptide and GST-Nogo-66. Binding persisted even after phosphatidylinositol- specific phospholipase C (PI-PLC) removal of GPI-linked F3 from the cell surface, suggesting a direct interaction between Nogo-66 and Caspr. Both Nogo-A and Caspr co-immunoprecipitated with Kv1.1 and Kv1.2, and the developmental expression pattern of both paralleled compared with Kv1.1, implicating a transient interaction between Nogo-A-Caspr and K(+) channels at early stages of myelination. In pathological models that display paranodal junctional defects (EAE rats, and Shiverer and CGT(-/-) mice), distances between the paired labeling of K(+) channels were shortened significantly and their localization shifted toward paranodes, while paranodal Nogo-A congregation was markedly reduced. Our results demonstrate that Nogo-A interacts in trans with axonal Caspr at CNS paranodes, an interaction that may have a role in modulating axon-glial junction architecture and possibly K(+)-channel localization during development.

L3 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:191844 BIOSIS
DOCUMENT NUMBER: PREV200400190218
TITLE: On the molecular architecture of axoglial junction.
AUTHOR(S): Xiao, Z.-C. [Reprint Author]
CORPORATE SOURCE: Department of Clinical Research, Singapore General Hospital, Outram Road, Singapore, Singapore
SOURCE: Journal of Neurochemistry, (February 2004) Vol. 88, No. Supplement 1, pp. 15. print.
Meeting Info.: 6th Biennial Meeting of the Asian-Pacific Society for Neurochemistry (APSN). Hong Kong, China. February 04-07, 2004. Asian-Pacific Society for Neurochemistry.
CODEN: JONRA9. ISSN: 0022-3042.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2004
Last Updated on STN: 7 Apr 2004

L3 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:43055 BIOSIS

DOCUMENT NUMBER: PREV200400044088
TITLE: Nogo-A at CNS paranodes is a ligand of Caspr: Possible regulation of K+ channel localization.
AUTHOR(S): Nie, Du-Yu; Zhou, Zhi-Hong; Ang, Beng-Ti; Teng, Felicia Y. H.; Xu, Gang; Xiang, Tao; Wang, Chao-yang; Zeng, Li; Takeda, Yasuo; Xu, Tian-Le; Ng, Yee-Kong; Faivre-Sarrailh, Catherine; Popko, Brian; Ling, Eng-Ang; Schachner, Melitta; Watanabe, Kazutada; Pallen, Catherine J.; Tang, Bor Luen; Xiao, Zhi-cheng [Reprint Author]
CORPORATE SOURCE: Department of Clinical Research, Singapore General Hospital, Singapore, Singapore
cpallen@interchange.ubc.ca; mcbtbl@imcb.nus.edu.sg; gcrxzc@sgh.com.sg
SOURCE: EMBO (European Molecular Biology Organization) Journal, (November 3 2003) Vol. 22, No. 21, pp. 5666-5678. print.
ISSN: 0261-4189 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 2004
Last Updated on STN: 14 Jan 2004
AB We report Nogo-A as an oligodendroglial component congregating and interacting with the Caspr-F3 complex at paranodes. However, its receptor Nogo-66 receptor (NgR) does not segregate to specific axonal domains. CHO cells cotransfected with Caspr and F3, but not with F3 alone, bound specifically to substrates coated with Nogo-66 peptide and GST-Nogo-66. Binding persisted even after phosphatidylinositol-specific phospholipase C (PI-PLC) removal of GPI-linked F3 from the cell surface, suggesting a direct interaction between Nogo-66 and Caspr. Both Nogo-A and Caspr co-immunoprecipitated with Kv1.1 and Kv1.2, and the developmental expression pattern of both paralleled compared with Kv1.1, implicating a transient interaction between Nogo-A-Caspr and K+ channels at early stages of myelination. In pathological models that display paranodal junctional defects (EAE rats, and Shiverer and CGT-/- mice), distances between the paired labeling of K+ channels were shortened significantly and their localization shifted toward paranodes, while paranodal Nogo-A congregation was markedly reduced. Our results demonstrate that Nogo-A interacts in trans with axonal Caspr at CNS paranodes, an interaction that may have a role in modulating axon-glial junction architecture and possibly K+-channel localization during development.

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FILE LAST UPDATED: 10 Dec 2006  (20061210/ED)

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=> S (E3)
L1            3 ("XIAO ZHI CHENG"/IN)

=> DIS L1 1 IBIB IABS
THE ESTIMATED COST FOR THIS REQUEST IS 2.74 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:515540  CAPLUS
DOCUMENT NUMBER:          141:65120
TITLE:                    Central nervous system damage
INVENTOR(S):              Xiao, Zhi-cheng
PATENT ASSIGNEE(S):       Singapore General Hospital Pte Ltd., Singapore;
```

Denison, Christopher M.
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004052922	A2	20040624	WO 2003-GB5323	20031205
WO 2004052922	A3	20041028		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2508847	AA	20040624	CA 2003-2508847	20031205
AU 2003288434	A1	20040630	AU 2003-288434	20031205
EP 1567545	A2	20050831	EP 2003-780353	20031205
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1745094	A	20060308	CN 2003-80109532	20031205
JP 2006526382	T2	20061124	JP 2004-558794	20031205
US 2006205668	A1	20060914	US 2005-537648	20050606
PRIORITY APPLN. INFO.:			US 2002-431620P P	20021206
			WO 2003-GB5323 W	20031205

ABSTRACT:
The application provides peptides that interact with the inhibitory domains of the myelin proteins Nogo, TNR and MAG. These may be used in the treatment for CNS damage, and for the development of further treatments. Also provided are methods and materials for immunizing subjects against the inhibitory domains of the myelin proteins, for the treatment for CNS damage.

=> DIS L1 2 IBIB IABS
THE ESTIMATED COST FOR THIS REQUEST IS 2.74 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:513552 CAPLUS
DOCUMENT NUMBER: 141:76737
TITLE: Materials and methods relating to treatment of injury and disease to the central nervous system
INVENTOR(S): Xiao, Zhi-cheng
PATENT ASSIGNEE(S): Singapore General Hospital Pte Ltd., Singapore; Forrest, Graham R.
SOURCE: PCT Int. Appl., 202 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004052389	A2	20040624	WO 2003-GB5329	20031205
WO 2004052389	A3	20041021		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2508715	AA	20040624	CA 2003-2508715		20031205
AU 2003295089	A1	20040630	AU 2003-295089		20031205
EP 1567186	A2	20050831	EP 2003-786089		20031205
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
CN 1744911	A	20060308	CN 2003-80109565		20031205
JP 2006516965	T2	20060713	JP 2005-502332		20031205
US 2006122110	A1	20060608	US 2005-537757		20050606
PRIORITY APPLN. INFO.:			US 2002-431549P	P	20021206
			US 2003-480138P	P	20030620
			WO 2003-GB5329	W	20031205

ABSTRACT:
The application provides materials and methods for promoting myelination of neuronal axons in the CNS. These derive from the findings firstly that the mols. Nogo and Caspr interact with one another during establishment and maintenance of the axoglial junction, and secondly that the mols. F3 and NB-3 are capable of promoting oligodendrocyte maturation via interaction with Notch. The materials and methods provided may be used in the treatment of CNS damage, in particular the treatment of spinal cord injury, multiple sclerosis, epilepsy and stroke.

=> DIS L1 3 IBIB IABS
THE ESTIMATED COST FOR THIS REQUEST IS 2.74 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:	1998:352868 CAPLUS
DOCUMENT NUMBER:	129:40126
TITLE:	Neuron and neural tumor growth regulatory system, antibodies thereto and uses thereof
INVENTOR(S):	Mckerracher, Lisa Joan; David, Samuel; Braun, Peter Erich; Xiao, Zhi-Cheng
PATENT ASSIGNEE(S):	Mckerracher, Lisa Joan, Can.; David, Samuel; Braun, Peter Erich; Xiao, Zhi-Cheng
SOURCE:	PCT Int. Appl., 79 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9822499	A2	19980528	WO 1997-CA868	19971117

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2190418	AA	19980515	CA 1996-2190418		19961115
CA 2221391	AA	19980515	CA 1997-2221391		19971117
AU 9850442	A1	19980610	AU 1998-50442		19971117
PRIORITY APPLN. INFO.:			CA 1996-2190418	A	19961115
			WO 1997-CA868	W	19971117

ABSTRACT:
The present invention relates to a neuron and neural tumor growth regulatory system, based on the novel protein, arretin and its isoforms and fragments thereof, its receptor, antibodies directed against the components of this system and diagnostic, therapeutic, and research uses for each of these aspects. This protein has an apparent mol. weight of approx. 70 kDa. Embodiments of the invention comprise the amino acid sequence and probes designed therefrom for nucleic acid sequences encoding arretin. Alternatively, tagged arretin protein for use as a reporter to detect receptors of arretin, which are then sequenced and used to obtain probes for the nucleic acid sequences encoding arretin receptors, are included. The present invention further relates to arretin receptors and fragments thereof as well as the nucleic acid sequences coding for such arretin receptors and fragments, and their therapeutic and diagnostic uses. Substances which function as either agonists or antagonists to arretin receptors are also envisioned and included within the scope of the present invention.

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WEST Search History

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DATE: Monday, December 11, 2006

Hide?	Set Name	Query	Hit Count
	DB=DWPI,JPAB,EPAB,USOC,USPT,PGPB; PLUR=YES; OP=ADJ		
☐	L5	XIAO-ZHI-CHENG!	6
☐	L4	XIAO-ZHI-CHENG!	6
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ		
☐	L3	L2 and l1	7
☐	L2	caspr	152
☐	L1	nogo	766

END OF SEARCH HISTORY

Case # 10,537,757
WEST (DWPI, JPAB, EPAB, USOC, USPT, PGPB)
12/11/06, AD